rejection is respectfully traversed on the basis that for it to be proper, at least inherency must be certain.

This burden is not satisfied, as the reference does not establish that inhibition of T cell destruction could be alleviated using a gp39 antagonist following the teachings of the references. Also, this reference relates to administration of a gp39 antagonist and cells that express an antigen to which T cell tolerance is to be achieved.

By contrast, the present claims are directed to inhibition of T cell mediated tissue destruction using a gp39 antagonist.

Claims 1-2, 5-10 and 12 stand rejected under 35 U.S.C. §102(e) as being anticipated by Lederman et al. The Office Action notes that this reference teaches treatment of a variety of autoimmune diseases including rheumatoid arthritis, Myasthenia gravis, SLE, Grave's disease, ITP, and diabetes using anti-gp39 antibodies. However, it is clear from the patent that the recognized mechanism of action of their discloses gp39 antibody therapies involves suppression of humoral immunity. Likewise this patent fails to teach or suggest inhibition of T cell mediated autoimmune diseases using a gp39 antagonist.

Again the Examiner indicates that the rejection is appropriate on inherency grounds. However, as with Noelle, this patent provides no basis for assuming that inhibition of T cell associated tissue destruction would be inhibited if the teachings of the reference are followed. For example, it cannot be assumed that a dosage protocol that inhibits humoral immunity will be sufficient to suppress T cell associated tissue destruction.

Withdrawal of the §112 rejection based on Lederman et al. is respectfully requested.

Claims 1-2, 5-10 and 12 further stand rejected 35 USC §103(a) as being obvious over Lederman et al., Armitage et al. in view of Schieven. This rejection is respectfully traversed on the basis that all of the cited references relate to the use of anti-gp39 antibodies to inhibit humoral immunity, i.e., antibody production. None of these references suggest that a gp39 antagonist could be used to suppress T cell mediated tissue destruction as claimed.

Again, Applications respectfully note that an inherency-based rejection requires that inherency be certain, not merely a possibility. Herein, there is no reason to conclude that practicing the methods disclosed in the cited references would necessarily suppress T cell

associated tissue destruction as this result will depend on factors such as the dosage of gp39 antagonist, disease status, and the particular disease treated.

Withdrawal of the §103 rejection based on Lederman et al., Armitage et al. and Schieven is therefore respectfully requested.

At the very least claims 18-26 should be free of the outstanding prior art rejection, as the diseases treated are not mentioned in the prior art.

Based on the foregoing, this application should be in order for allowance. A Notice to that effect is respectfully solicited.

Respectfully submitted,

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APPENDIX

--13. A method for inhibiting T cell mediated tissue destruction associated with an autoimmune disease other than multiple sclerosis comprising administering an effective amount of a gp39 antagonist selected from the group consisting of soluble CD40, a CD40 fusion protein, an anti-gp39, antibody or a fragment thereof that binds gp39.

14. The method of claim 13 wherein the gp39 antagonist is an anti-gp39 antibody or fragment thereof that binds gp39.

15. The method of claim 14 wherein the antibody is a humanized antibody.

16. The method of claim 14 wherein the antibody is a chimeric antibody.

17. The method of claim 13 wherein the gp39 antagonist is soluble CD40 or a CD40 fusion protein.

18. A method for treating oophoritis in a subject in need of such treatment comprising administering a therapeutically effective amount of a gp39 antagonist selected from the group consisting of soluble CD40, CD40 fusion protein and an anti-gp39 antibody or fragment thereof that binds gp39.

19. The method of claim 18 wherein said gp39 antagonist is an anti-gp39 antibody or fragment thereof that binds gp39.

20. The method of claim 19 wherein said antibody is a humanized antibody.

21. The method of claim 19 wherein said antibody is a chimeric antibody.

22. A method for treating thyroluitis comprising administering a therapeutically effective amount of a gp39 antagonist selected from the group consisting of an anti-gp39 antibody or fragment thereof that binds gp39, soluble CD40 and a CD40 fusion protein.

23. The method of claim 22 wherein said gp39 antagonist is an anti-gp39 antibody or fragment thereof that binds gp39.

24. The method of claim 23 wherein said antibody is a humanized antibody.

25. The method of claim 23 wherein said antibody is a chimerized antibody.

26. The method of claim 22 wherein said gp39 antagonist is soluble CD40 or CD40 fusion protein.